Bicarbonate Dialysis

Prof Nagy Sayed-Ahmed

1. Definition & Stages of CKD
2. CKD & ESRD magnitude of the problem in EGYPT, USA & WW
3. Uremic syndrome & Uremic Toxins
4. Acidosis and Uremia
5. Methods of treatment of Uremia
6. Extracorporeal Purification Methods
7. History of Hemodialysis
8. Dialysate Composition and its role in patients' well being
9. Dialysate Buffers and role of dialysis for treatment of in treatment of uremic acidosis

10. Evolution from bicarbonate to Acetate to Bicarbonate
11. Disadvantages of Acetate dialysis
12. Advantages of Bicarbonate dialysis
13. Studies in favor of bicarbonate dialysis
14. Conversion from Acetate to Bicarbonate in our locality
15. Different Types of Bicarbonate dialysis
16. Disadvantages of Bicarbonate dialysis
17. Cost differences
18. Searching for a better dialysate buffer
19. Take home message

Table 10. Stages of Chronic Kidney Disease

Stage	Description	GFR (mL/min/1.73 m^2)
1	Kidney damage with normal or ↑ GFR	≥90
2	Kidney damage with mild ↓ GFR	60–89
3	Moderate ↓ GFR	30–59
4	Severe ↓ GFR	15–29
5	Kidney failure	<15 (or dialysis)

Chronic kidney disease is defined as either kidney damage or GFR <60 mL/min/1.73 m^2 for ≥3 months. Kidney damage is defined as pathologic abnormalities or markers of damage, including abnormalities in blood or urine tests or imaging studies.

ON

GRANULAR DEGENERATION

OF

THE KIDNIES,

AND ITS CONNEXION WITH

DROPSY, INFLAMMATIONS, AND OTHER DISEASES.

BY

ROBERT CHRISTISON, M.D. F.R.S.E.

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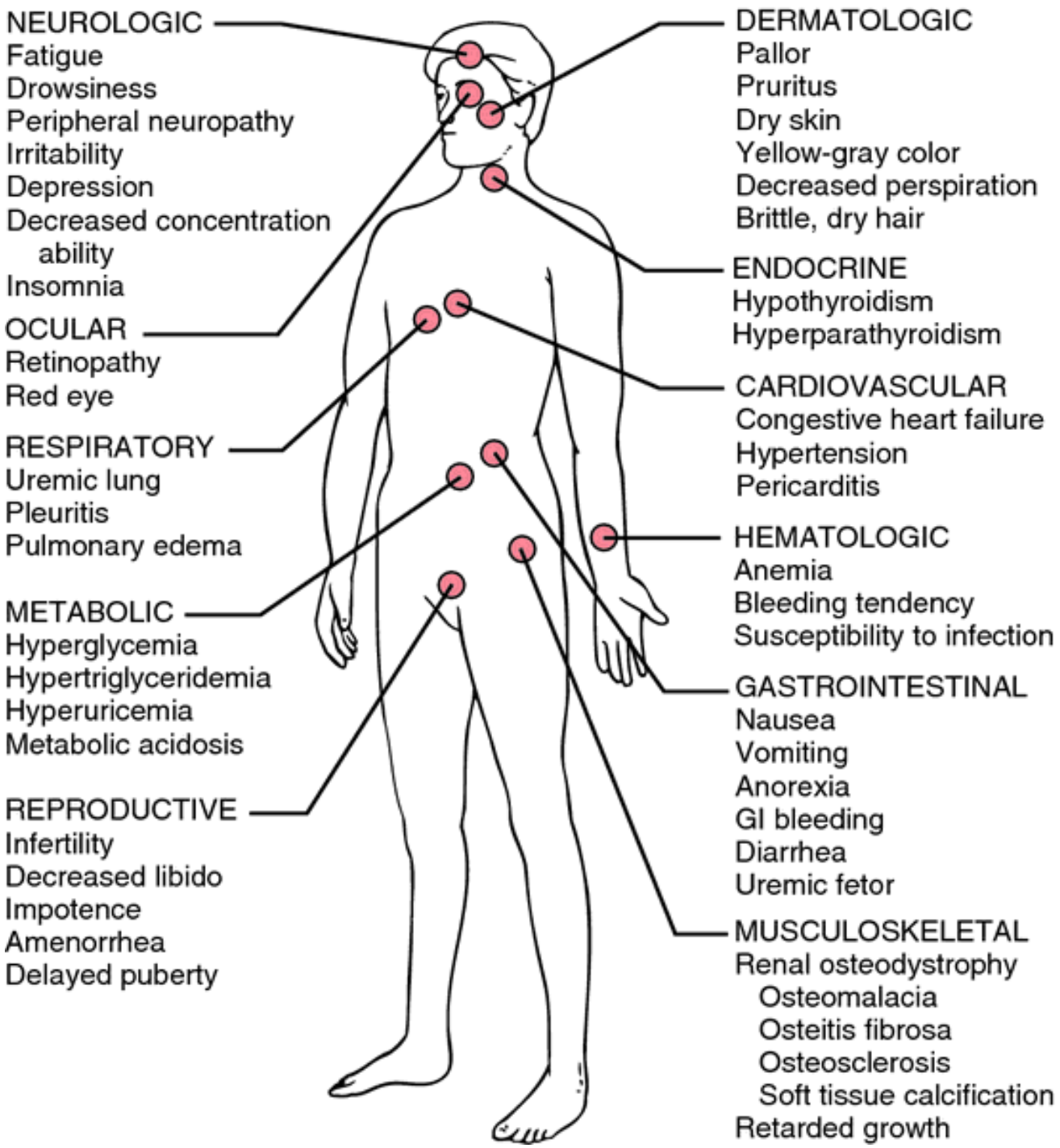
EDINBURGH:

ADAM AND CHARLES BLACK, NORTH BRIDGE:

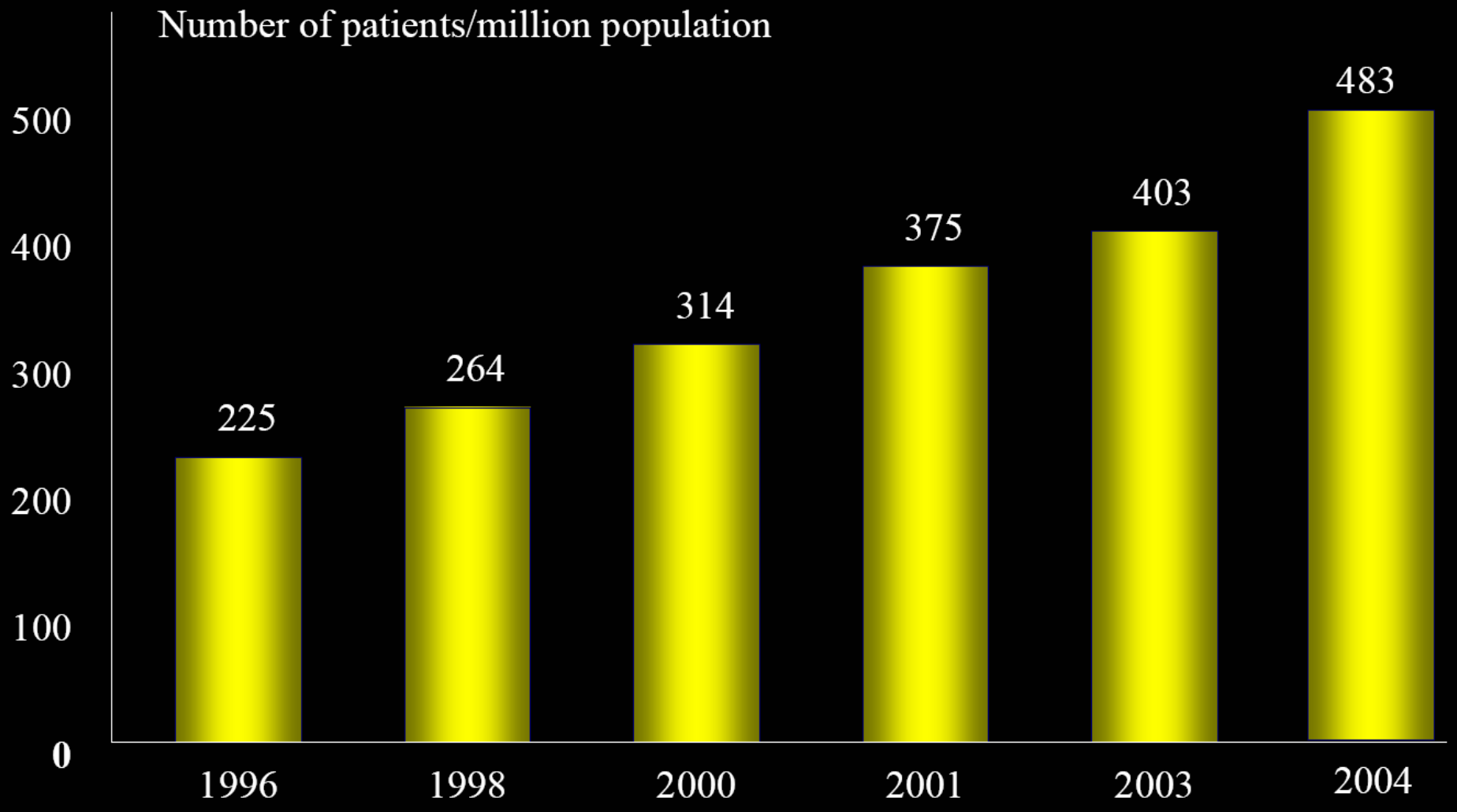
AND LONGMAN, ORME, BROWN, GREEN, AND LONGMANS, LONDON.

MDCCCXXXIX.

(Sir) Robert Christison (1797–1882) of Edinburgh, Scotland (a), who first articulated in 1839 a theory of uraemia based on the retention of solutes, particularly urea


NEUROLOGIC
Fatigue
Drowsiness
Peripheral neuropathy
Irritability
Depression
Decreased concentration ability
Insomnia
OCULAR
Retinopathy
Red eye
RESPIRATORY
Uremic lung
Pleuritis
Pulmonary edema
METABOLIC
Hyperglycemia
Hypertriglyceridemia
Hyperuricemia
Metabolic acidosis
REPRODUCTIVE
Infertility
Decreased libido
Impotence
Amenorrhea
Delayed puberty
DERMATOLOGIC
Pallor
Pruritus
Dry skin
Yellow-gray color
Decreased perspiration
Brittle, dry hair
ENDOCRINE
Hypothyroidism
Hyperparathyroidism
CARDIOVASCULAR
Congestive heart failure
Hypertension
Pericarditis
HEMATOLOGIC
Anemia
Bleeding tendency
Susceptibility to infection
GASTROINTESTINAL
Nausea
Vomiting
Anorexia
GI bleeding
Diarrhea
Uremic fetor
MUSCULOSKELETAL
Renal osteodystrophy
Osteomalacia
Osteitis fibrosa
Osteosclerosis
Soft tissue calcification
Retarded growth

Prevalence of ESRD in Egypt



Adel Affifi: The Egyptian Renal Registry 9th Annual Report 2008

SOME UREMIC TOXINS - 1

- By-products of ptn & AA metabolism:
  - Urea
  - Guanidino compounds:
    - Creatinine & creatine & sarcosine
    - Guanidine
    - Methyl & dimethyl guanidine
    - Guanidinosuccinic acid
  - End products of nucleic acid metabolism:
    - Urates & hippurates
  - End products of aliphatic & aromatic AA metabolism
  - Other nitrogenous substances
  - Advanced glycation end products
- Inhibitos of ligand-protein binding & inhibitor of somatomedin and insulin action
- Middle molecules (500-12000 Da)

UREMIC TOXINS - 2

- Urea → anorexia, malaise, nausea, vomiting
- Guanidinosuccinic acid → interfers with activation of platelet factor III by ADP
- PTH, insulin, glucagon, LH, prolactin: decreased degradation + enhanced secretion
- PTH: adverse effect of elevating cellular cytosolic Ca2+ levels in several tissues and organs

Acid-base Balance In Chronic Kidney Disease

- Acid-base balance is normally maintained by the renal excretion of the daily acid load (about 1 meq/kg per day, derived mostly from the generation of sulfuric acid during the metabolism of sulfur-containing amino acids)

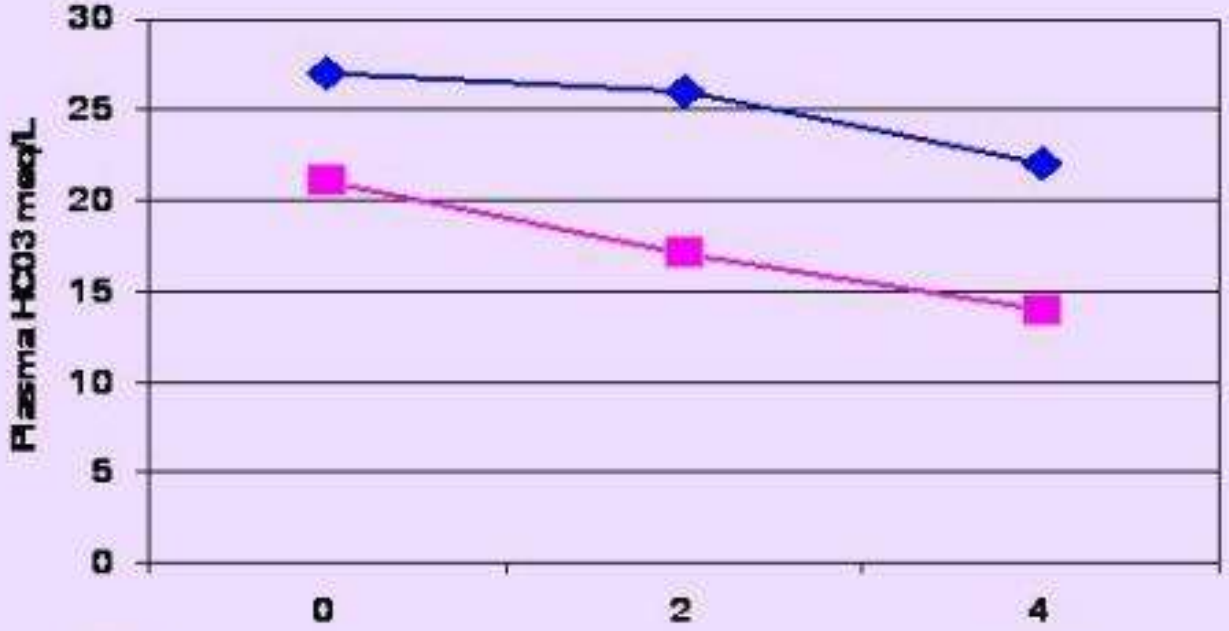
H^+ Secretion in CKD

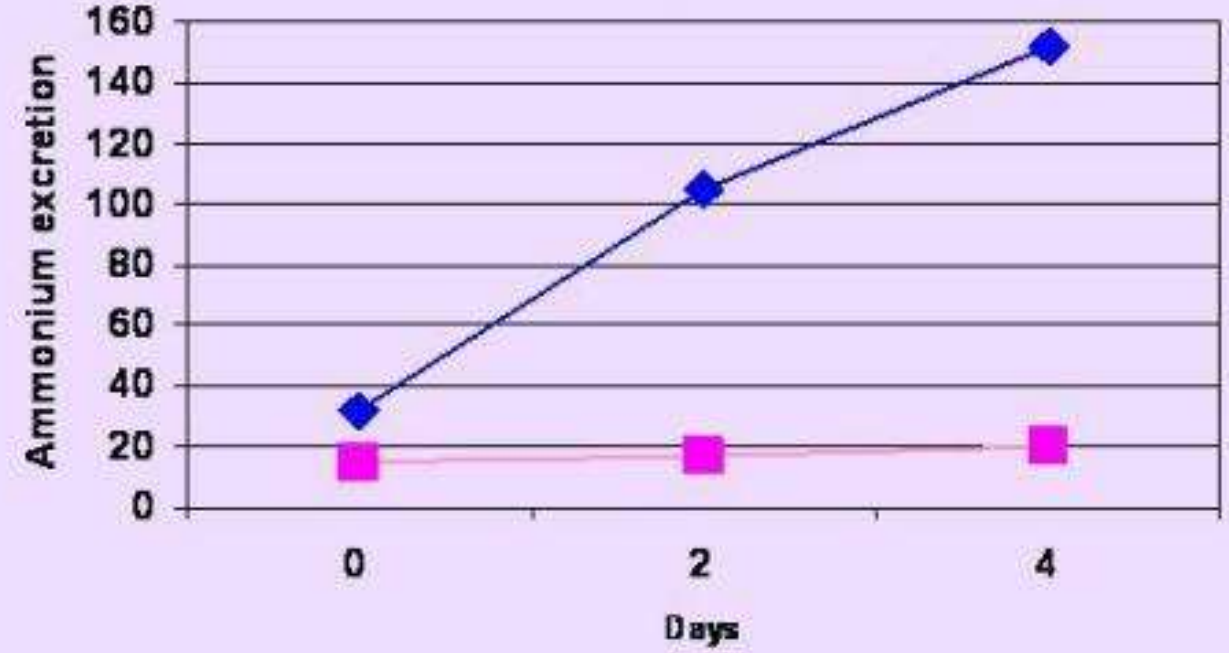
In early stages of CKD:

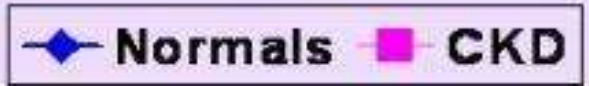
- H^+ balance and HCO_3^- remain normal due to increases in:
  - H^+ secretion per residual nephron
  - NH_3 (buffer) production increases

In late stages of CKD

- Metabolic acidosis develops due to decreased:
  - NH_3 generation****
  - $HC0_3$ reabsorption
  - Excretion of hydrogen ions
- $HC0_3$ levels falls to 12 to 20 and then stabilizes due to bone buffering







Metabolic acidosis in CKD

- a mixture of normal anion gap and increased anion gap;
- the kidneys are unable to produce enough ammonia in the proximal tubules to excrete the endogenous acid into the urine in the form of ammonium.
- accumulation of phosphates, sulfates, and other organic anions

Deleterious Effects Of Metabolic Acidosis

- Negative nitrogen balance
- Increased protein degradation
- Increased essential amino acid oxidation
- Reduced albumin synthesis
- Lack of adaptation to a low protein diet
- Renal osteodystrophy: bone acts as a buffer for excess acid, with resultant loss of mineral.
- Acidosis interferes with vitamin D metabolism

Replacement of Failed Organ Function

- The challenge of replacing or restoring missing body parts, diseased organs, or defective physiologic functions
- A functional prosthetic toe found on an egyptian mummy dated to approximately 1800 BC
- Glass eyes, wooden legs, and iron lungs
- Frequent injection of xenogeneic insulin to treat diabetes caused by exocrine pancreas failure

Organ Replacement Strategies

- from the late 1950s and early 1960s: surgeons + engineers introduced transplants and man-made organometallic devices – replace the function of
  - kidney,
  - portions of the heart,
  - the lung, and
  - large joints

Value of RRT

- By the end of the last millennium nearly one million individuals worldwide, who had suffered complete and irreversible failure of kidney function, were being maintained alive by a 'temporary' treatment: dialysis
- The kidney was the first organ for which complete mechanical substitution became possible

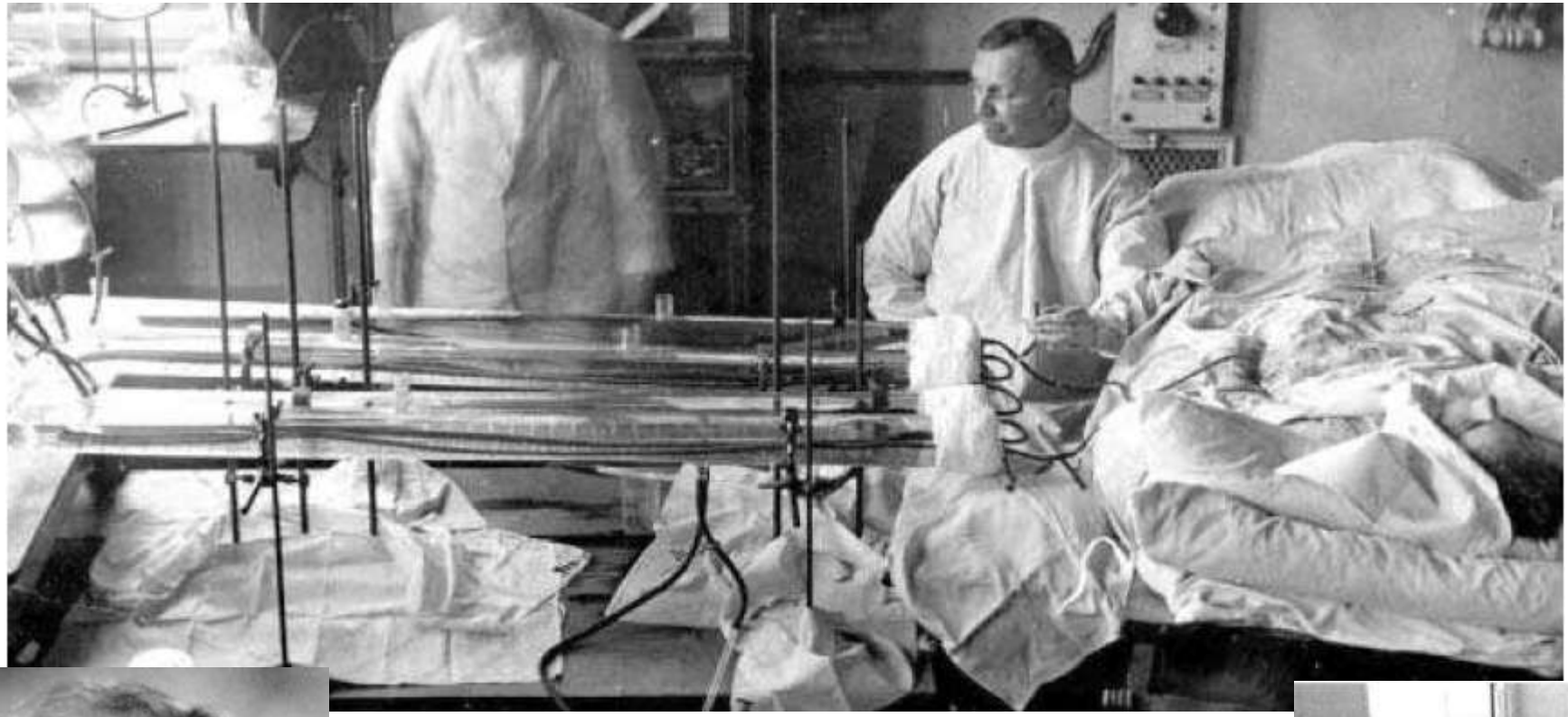
Dialysis

- Maintenance dialysis is the most remarkable contemporary approach to organ replacement
- > $1.5X10^6$ people in the world are alive just because they have access to one form or another of RRT
- 90% live in the developed countries (average gross income >$10 000 per capita)
- RRT is so costly: the vast majority of the world's population unable to take advantage of it

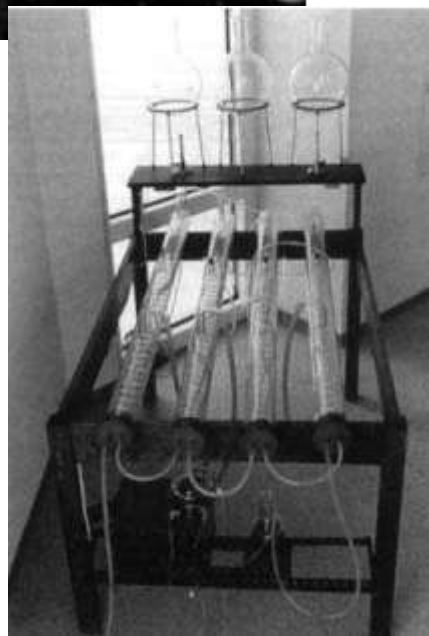
Hemodialysis

- Currently sustain or vastly improve the lives of >20 million recipients
- High-technology organ replacement accounts for ~ 8% of worldwide health care expenditure
- WW, costs of organ prosthesis exceeds $300 billion US dollars per year and represents between 7 and 8% of total worldwide health care spending.
- ??? ESRD maintenance therapy really represents the "highest and best" allocation of society's finite health care expenditure

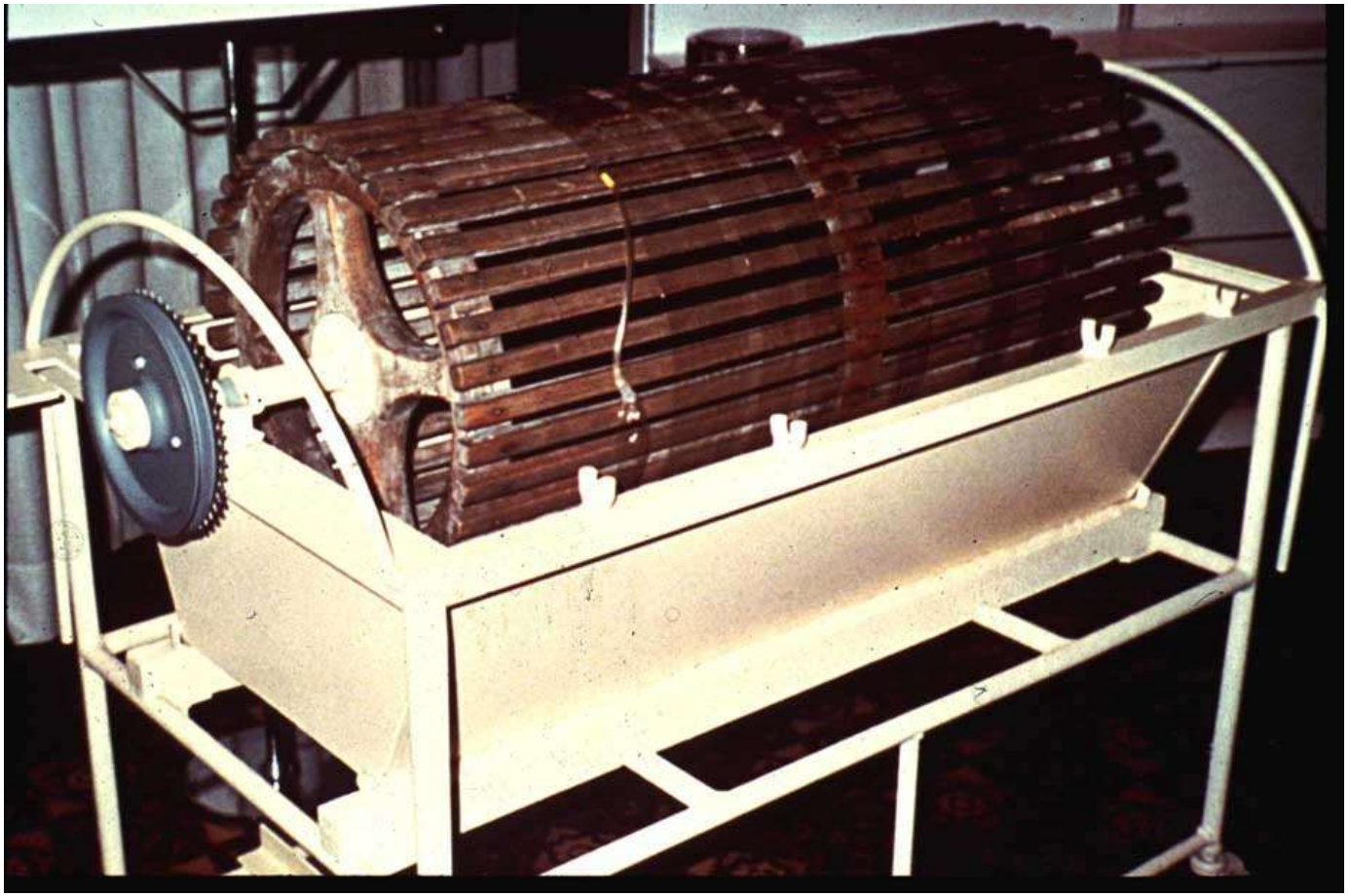
Thomas Graham (1805–1869) the ‘father’ of dialysis. Described diffusion, re-defined the word *dialysis,* distinguished what he named ‘crystalloids’from ‘colloids’, described the first ever dialysis membrane. ‘Molecules are moved by the force of diffusion’

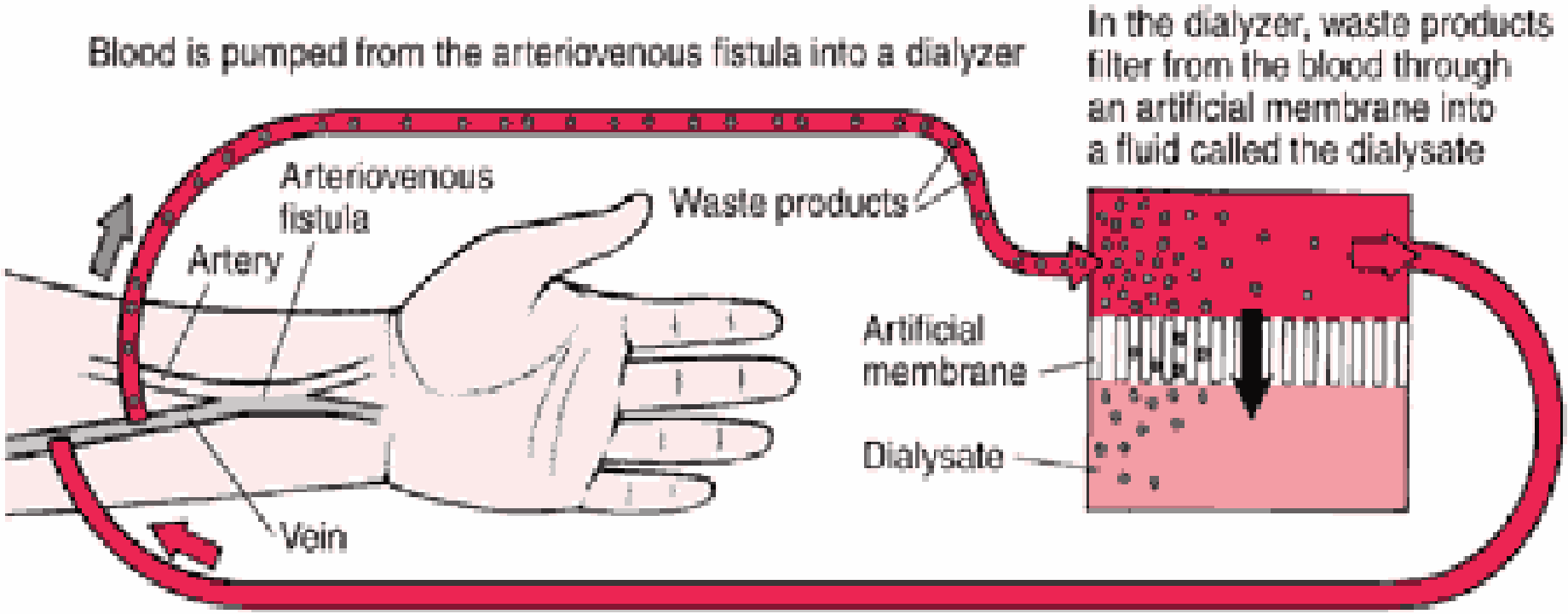


Haas performing a dialysis on a young girl in 1926



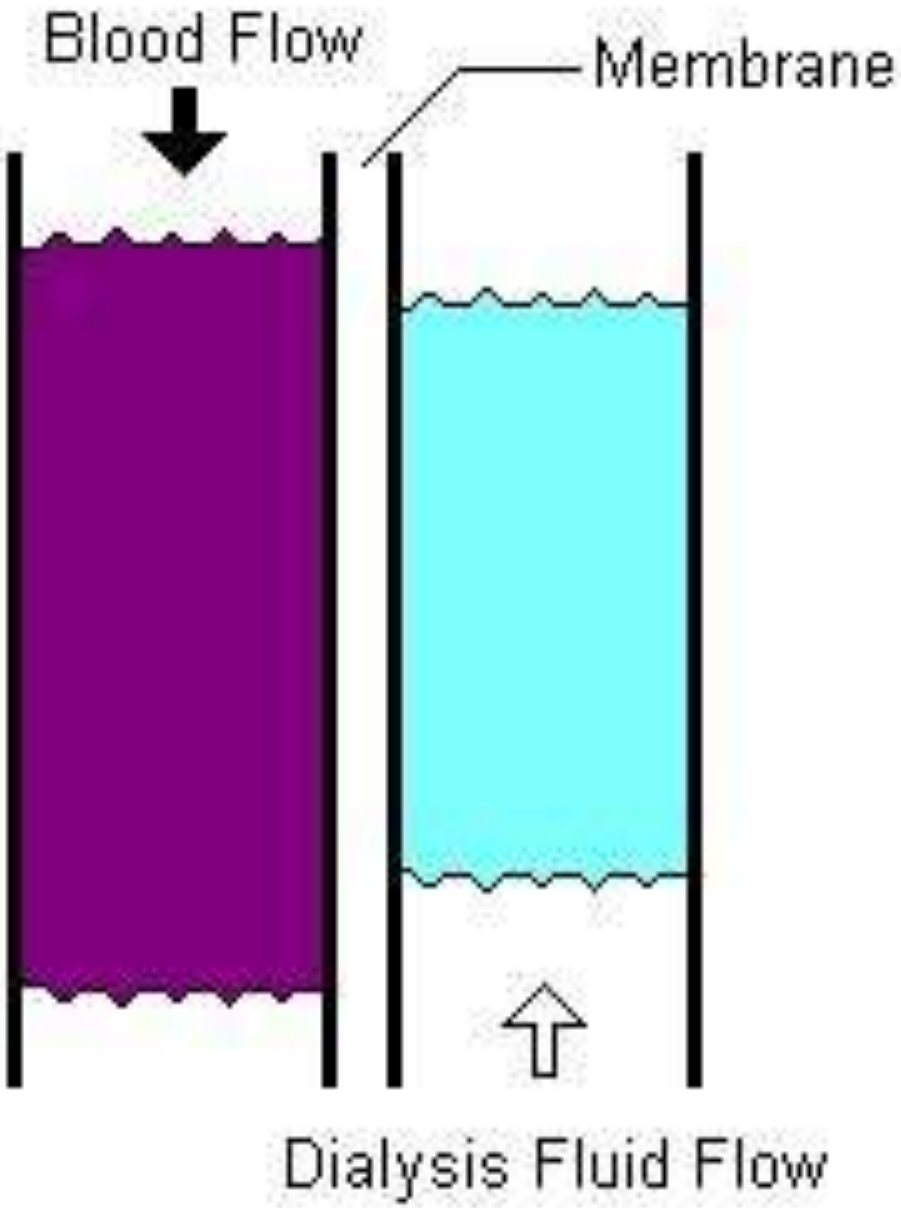
Dr. Willem Kolff is considered the father of dialysis. This young Dutch physician constructed the first dialyzer (artificial kidney) in 1943.

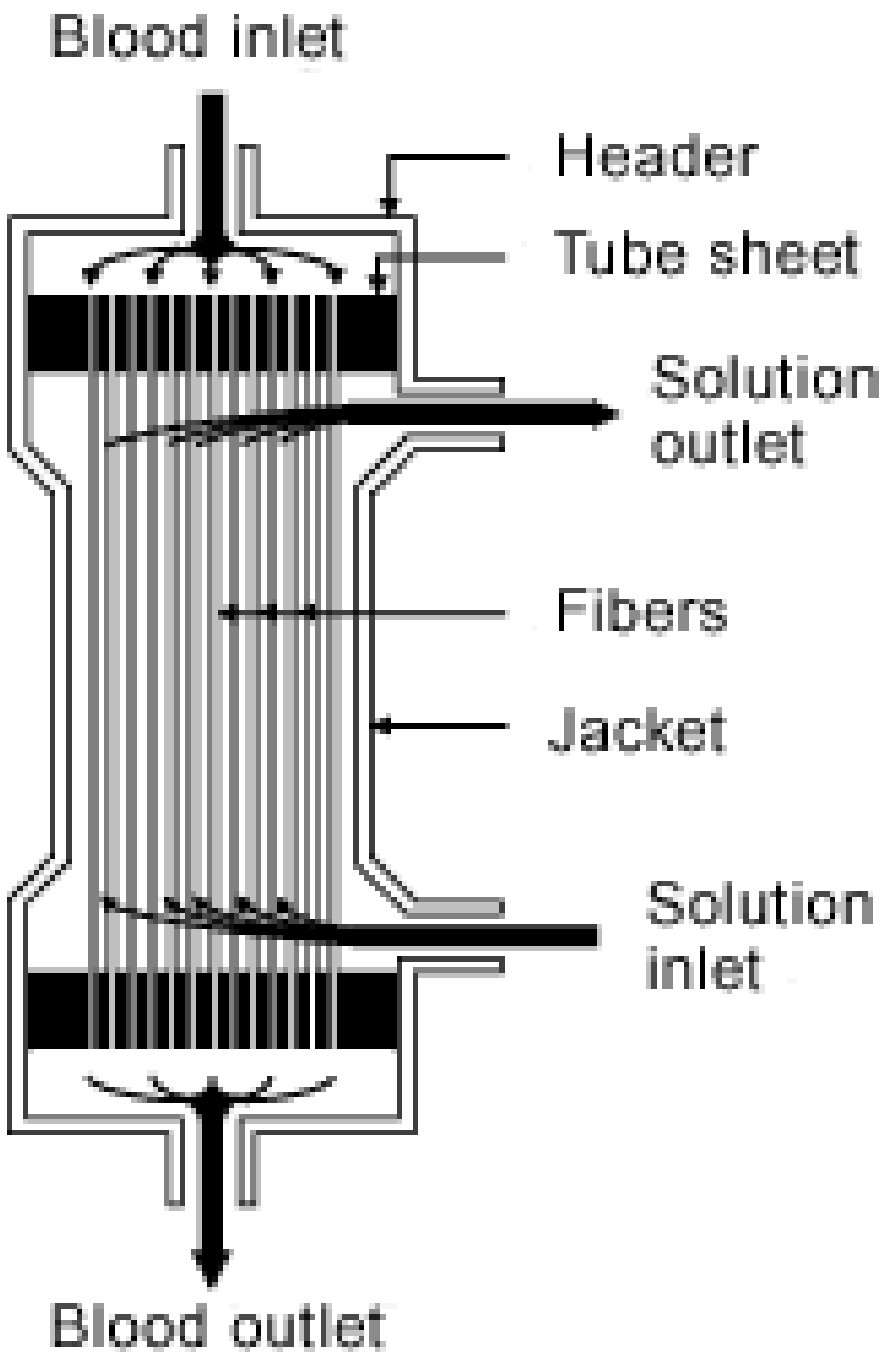


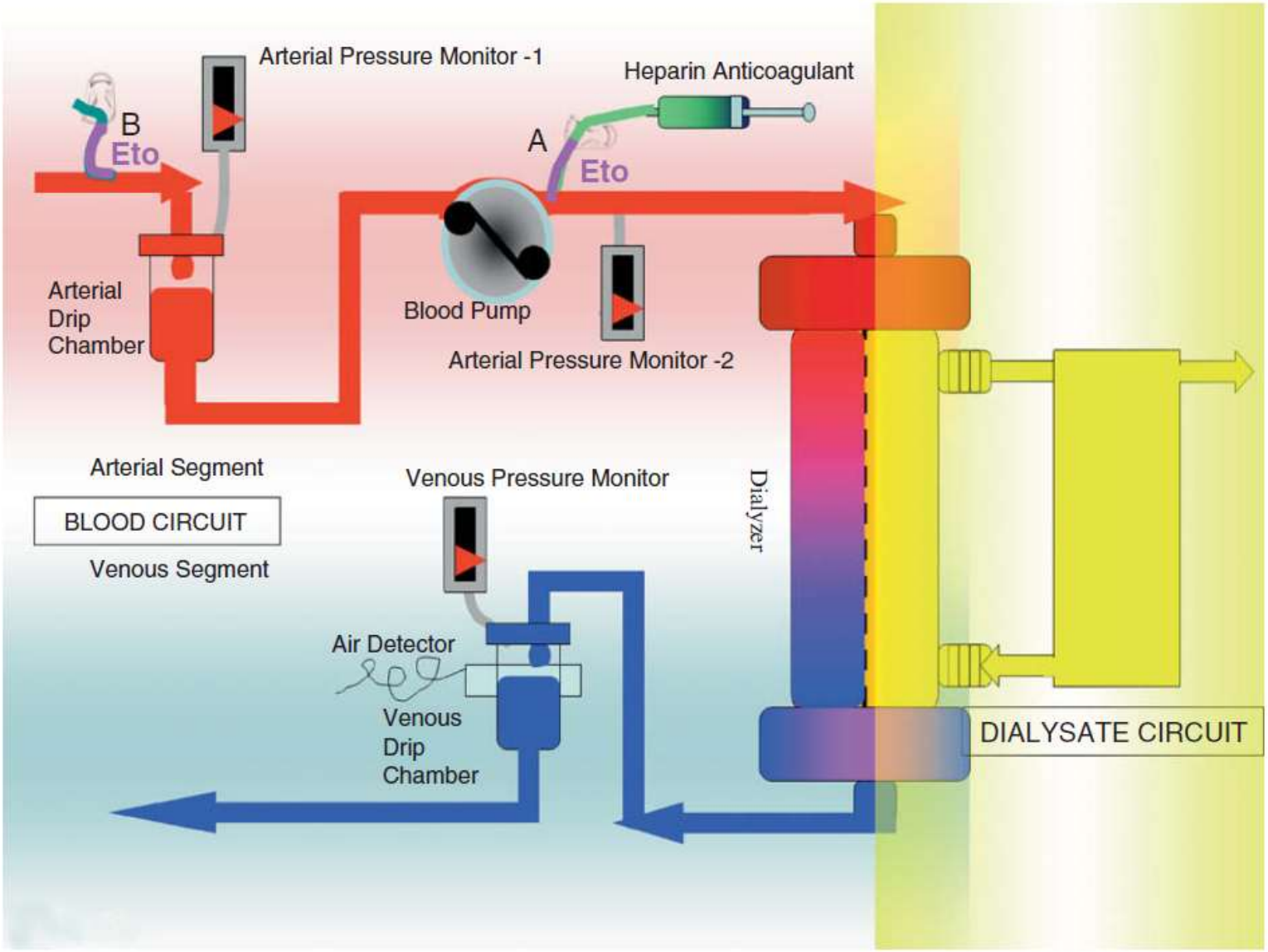


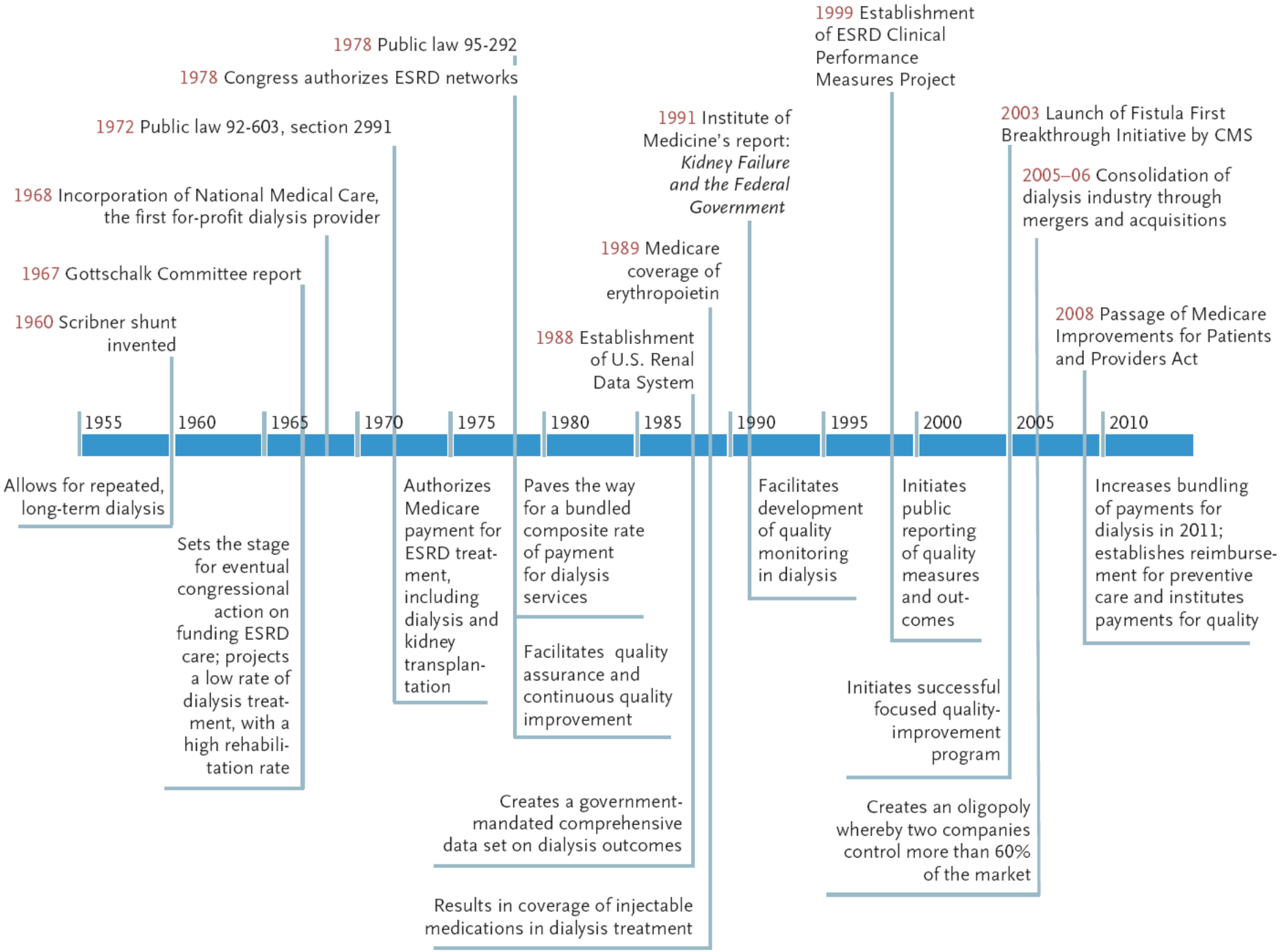
Hemodialysis

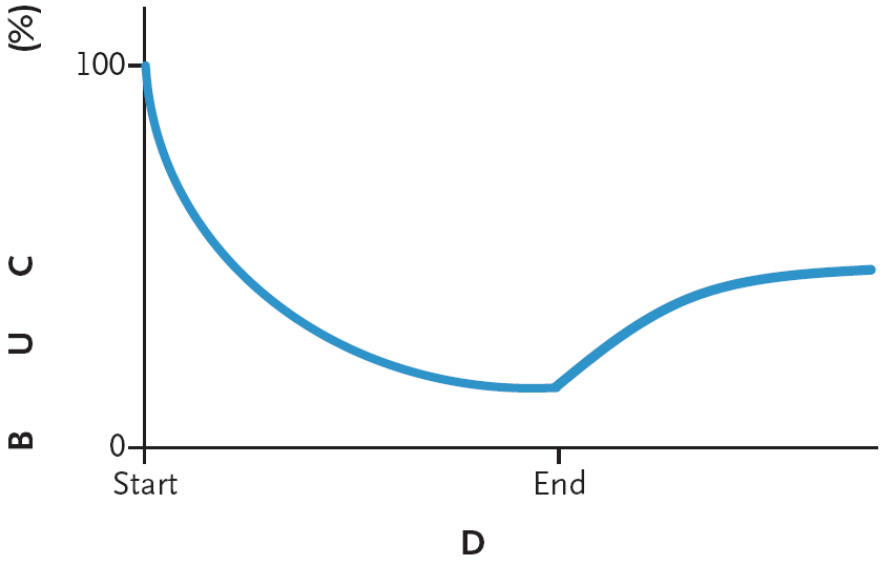
Countercurrent Flow

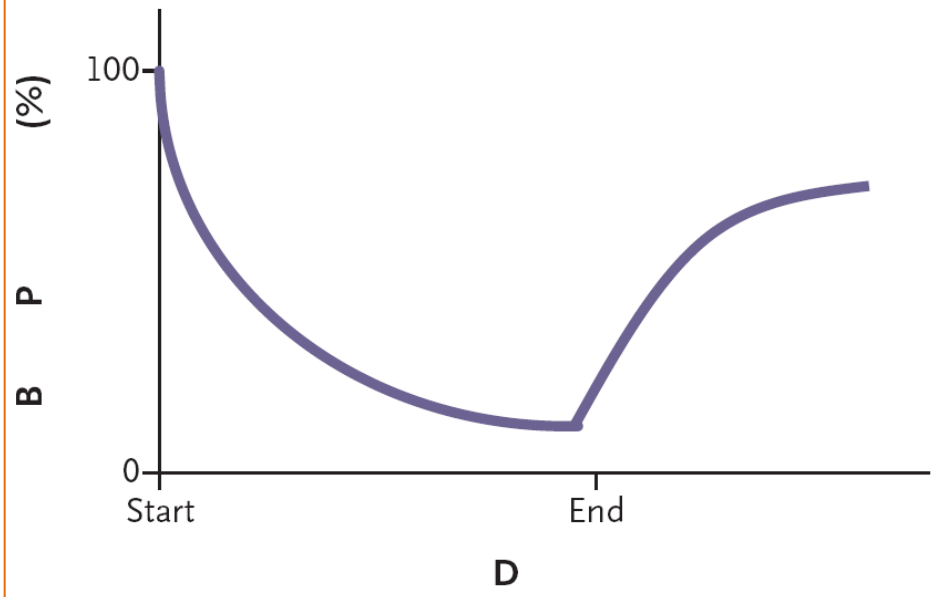


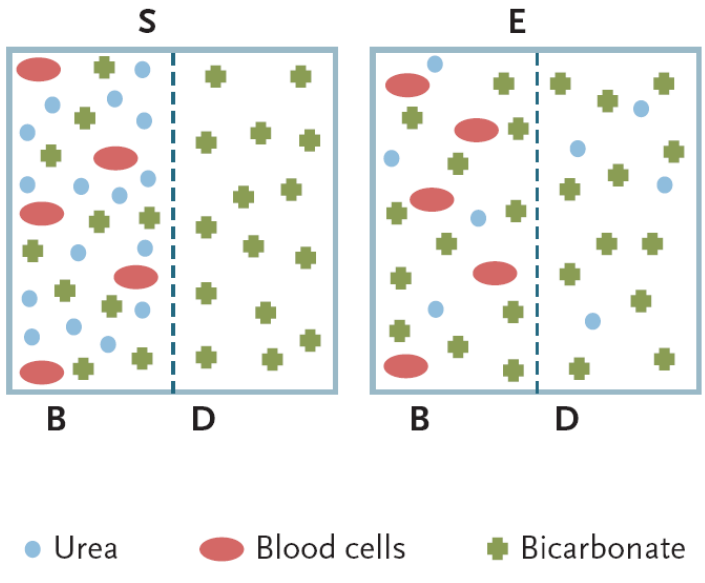

Blood inlet
Header
Tube sheet
Solution outlet
Fibers
Jacket
Solution inlet
Blood outlet

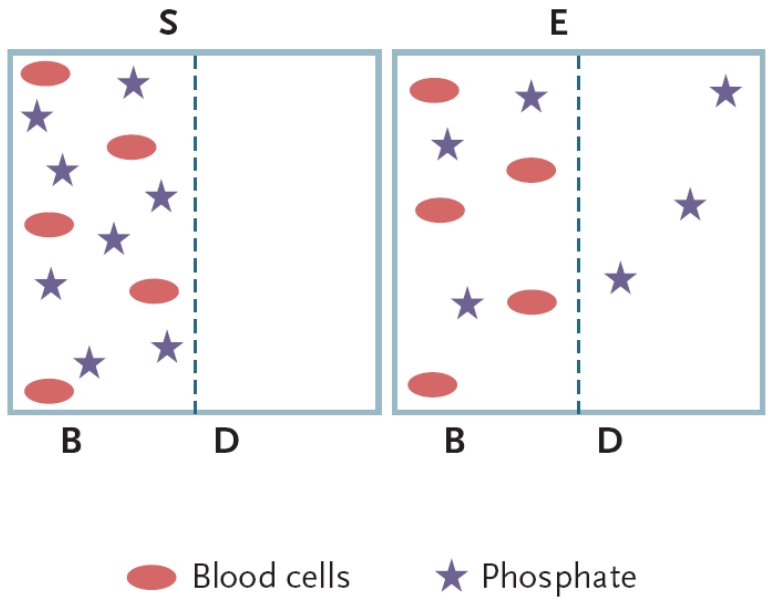

Arterial Pressure Monitor -1
Heparin Anticoagulant
B
Eto
A
Eto
Arterial
Drip
Chamber
Blood Pump
Arterial Pressure Monitor -2
Arterial Segment
BLOOD CIRCUIT
Venous Segment
Venous Pressure Monitor
Dialyzer
Air Detector
Venous
Drip
Chamber
DIALYSATE CIRCUIT


1960 Scribner shunt invented
Allows for repeated, long-term dialysis
1967 Gottschalk Committee report
Sets the stage for eventual congressional action on funding ESRD care; projects a low rate of dialysis treatment, with a high rehabilitation rate
1968 Incorporation of National Medical Care, the first for-profit dialysis provider
1972 Public law 92-603, section 2991
Authorizes Medicare payment for ESRD treatment, including dialysis and kidney transplantation
1978 Congress authorizes ESRD networks
1978 Public law 95-292
Paves the way for a bundled composite rate of payment for dialysis services
Facilitates quality assurance and continuous quality improvement
1988 Establishment of U.S. Renal Data System
1989 Medicare coverage of erythropoietin
Results in coverage of injectable medications in dialysis treatment
1991 Institute of Medicine's report: Kidney Failure and the Federal Government
Creates a government-mandated comprehensive data set on dialysis outcomes
1999 Establishment of ESRD Clinical Performance Measures Project
Facilitates development of quality monitoring in dialysis
Initiates public reporting of quality measures and outcomes
2003 Launch of Fistula First Breakthrough Initiative by CMS
Initiates successful focused quality-improvement program
2005–06 Consolidation of dialysis industry through mergers and acquisitions
Creates an oligopoly whereby two companies control more than 60% of the market
2008 Passage of Medicare Improvements for Patients and Providers Act
Increases bundling of payments for dialysis in 2011; establishes reimbursement for preventive care and institutes payments for quality
1955
1960
1965
1970
1975
1980
1985
1990
1995
2000
2005
2010


BUC (%)
100
0
Start
End
D


BP (%)
100
0
Start
End
D


S
E
B
D
B
D
Urea
Blood cells
Bicarbonate

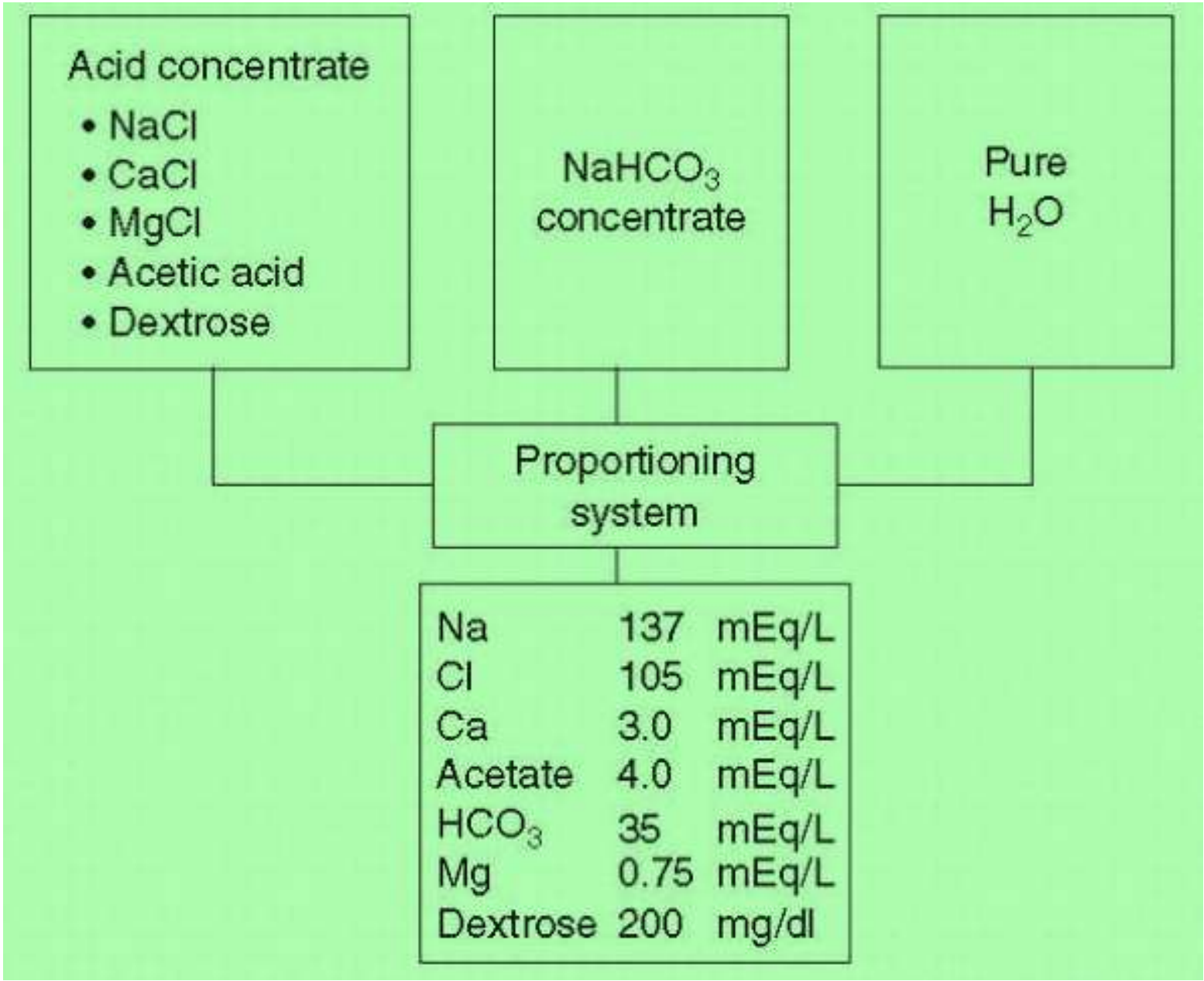

S
E
B
D
B
D
Blood cells
Phosphate

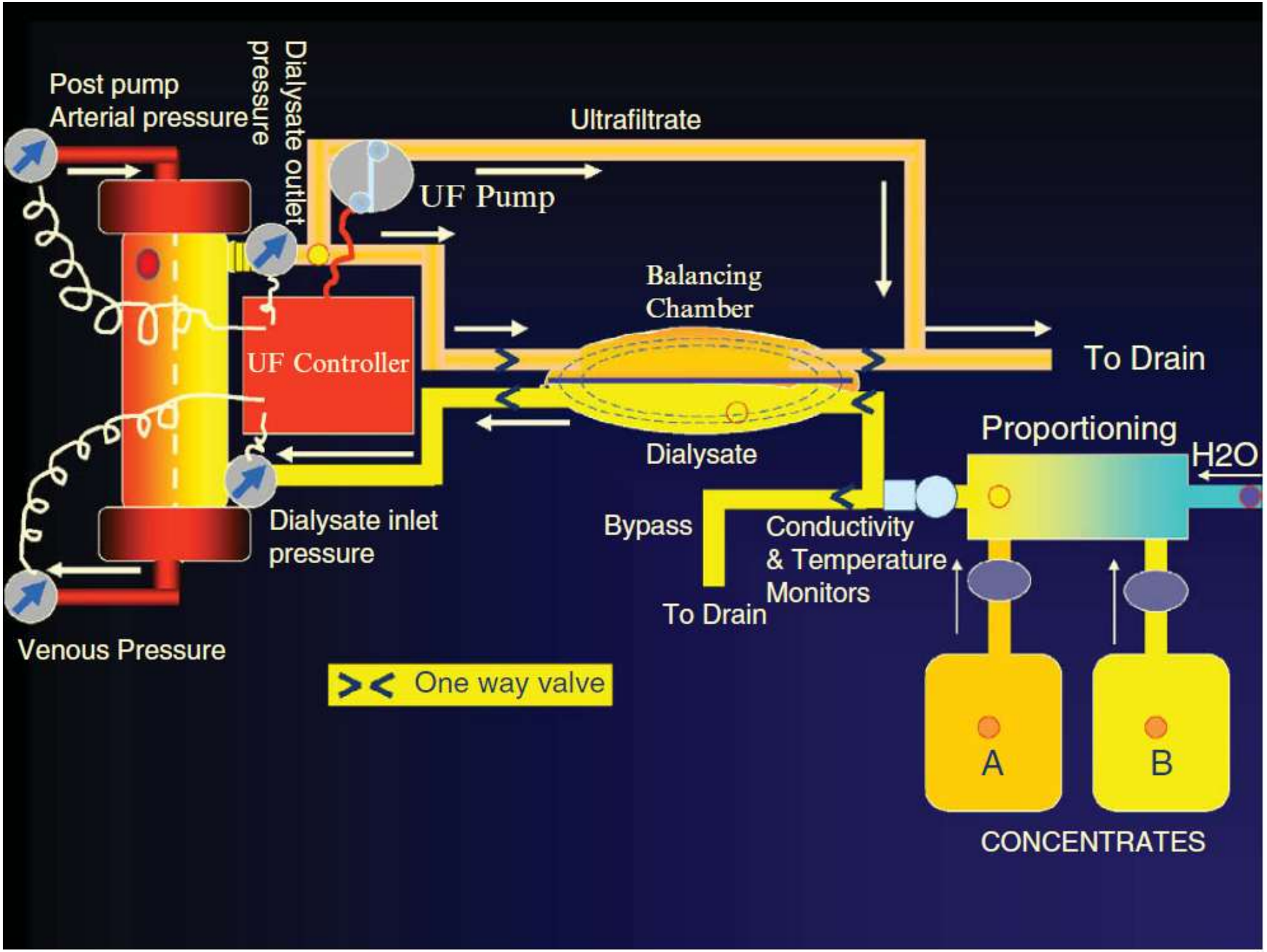
Dialysate composition

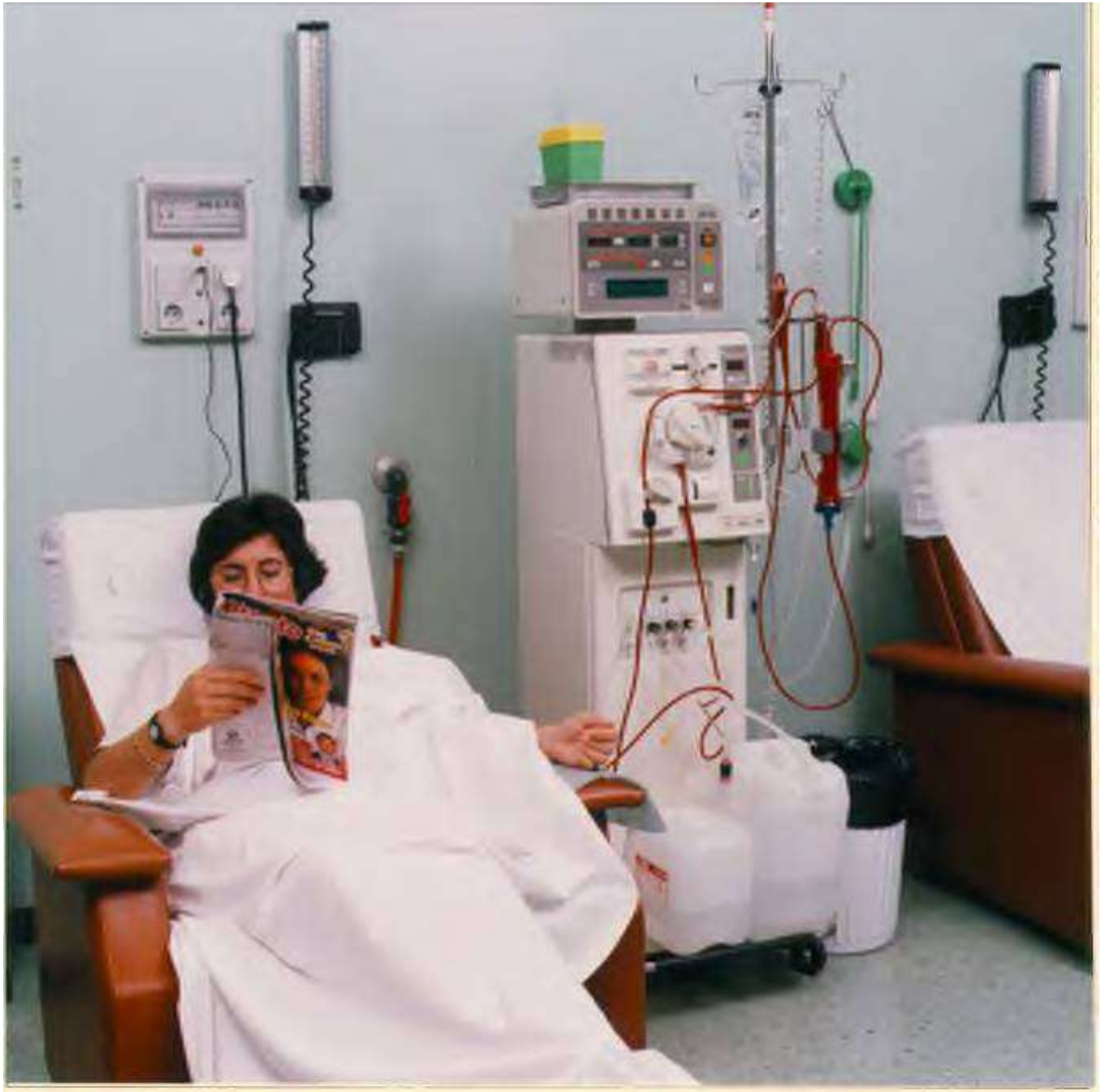
Sodium	Between 130 and 145 mmol per liter. Higher sodium concentrations decrease the risk of intradialytic hypotension but increase thirst and interdialytic weight gain.
Potassium	Generally 2 to 3 mmol per liter. Lower levels of dialysate potassium are associated with sudden cardiac death; intradialytic potassium removal is highly variable, and plasma potassium levels rebound about 30% after dialysis.
Calcium	Generally 1.25 to 1.75 mmol per liter. Only non–protein-bound calcium is removed; higher levels of dialysate calcium increase intradialytic blood pressure.
Magnesium	Generally 0.5 mmol per liter. The optimal level of magnesium is unresolved, and magnesium flux is difficult to predict.
Alkaline buffers	Commonly 30 to 40 mmol per liter. Predominantly bicarbonate with a small amount of acetate; bicarbonate concentration can be adjusted to correct metabolic acidosis.
Chloride	Defined by prescribed cations and alkaline buffers in dialysate.
Glucose	Commonly 100 to 200 mg per deciliter. Higher levels of glucose promote hypertriglyceridemia.

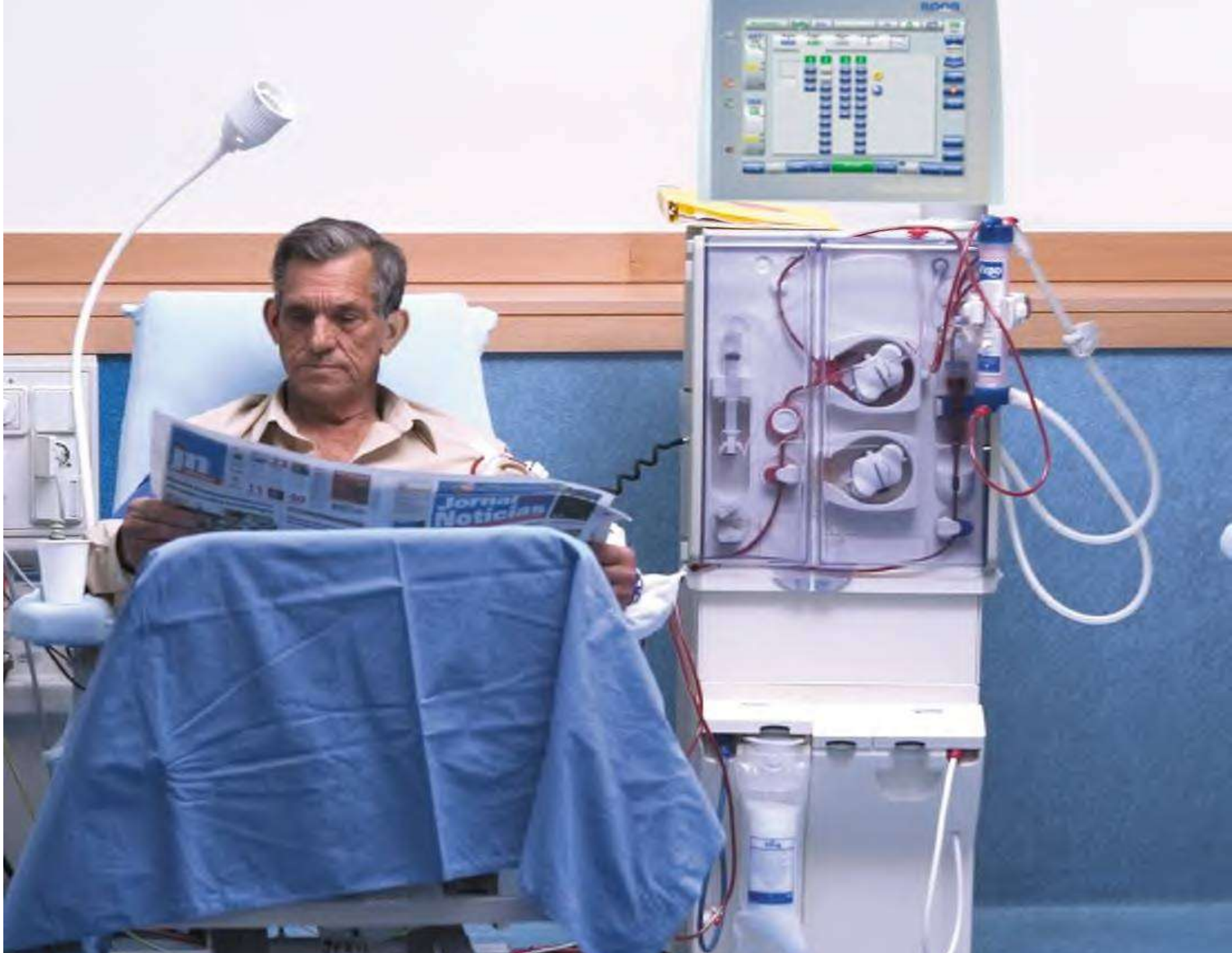
Composition of Dialysate (mEq/l)

Sodium	135 – 145
Potassium	0 – 4
Calcium	2.5 – 3.5
Magnesium	0.5 – 1.5
Chloride	98 – 112
Acetate / Citrate	4 – 10 / 2.4
Glucose	0 – 200 mg/dl
Bicarbonate	35 – 40


Acid concentrate
• NaCl
• CaCl
• MgCl
• Acetic acid
• Dextrose
NaHCO3 concentrate
Pure H2O
Proportioning system
Na 137 mEq/L
Cl 105 mEq/L
Ca 3.0 mEq/L
Acetate 4.0 mEq/L
HCO3 35 mEq/L
Mg 0.75 mEq/L
Dextrose 200 mg/dl

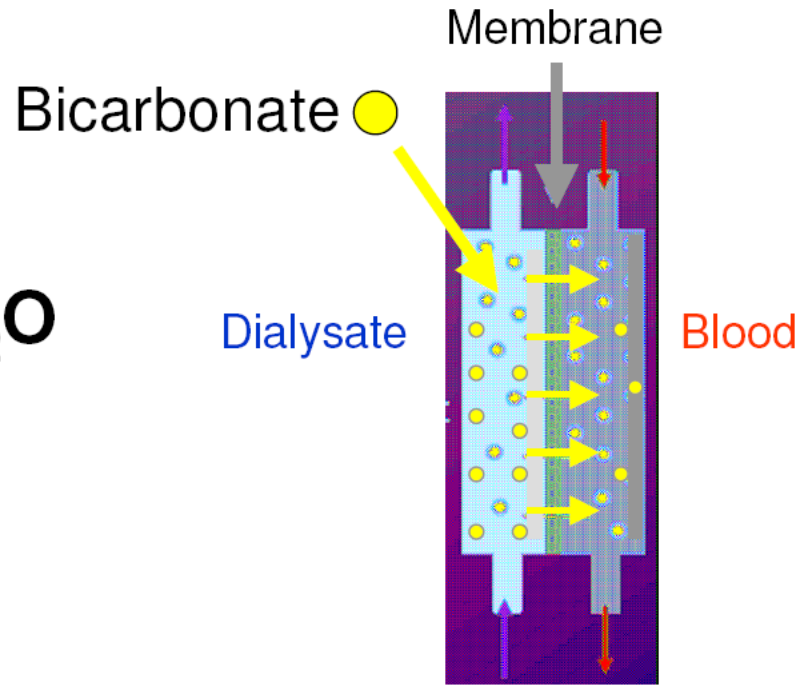

Post pump
Arterial pressure
Dialysate outlet pressure
Ultrafiltrate
UF Pump
Balancing Chamber
UF Controller
To Drain
Proportioning
H2O
Dialysate
Dialysate inlet pressure
Bypass
Conductivity & Temperature Monitors
To Drain
Venous Pressure
>< One way valve
A
B
CONCENTRATES




Jornal
Notícias

How do we correct metabolic acidosis ?

$$H^+ + HCO^-_3 = CO_2 + H_2O$$



Correction of acidosis:

→ **Dialysate bicarbonate conc. 30 – 40 mmol/l**

→ **Predialytic bicarbonat conc. > 20 mmol/l**

Zuchelli et al Kidney Int 43:S179;1993

Advantages and disadvantages of modifications in the dialysate composition

Substance		Advantage	Disadvantage
Na	↑	Hemodynamic stability	Thirst ↑ / weight gain ↑
	↓	Reduced osmotic stress	Hemodynamic stability ↓
K	↑	Arrhythmias ↓	Hyperkalemia
	↓	Dietary intake ↑	Risk of sudden death ↑
Ca	↑	PTH ↓	Hypercalcemia / Vascular calcification
	↓	Use of Ca-containing P-Binders ↑	Stimulation of PTH ↑
Mg	↑	PTH ↓ / Arrhythmias ↓	Nerve cond. / pruritus / bone disease
	↓	Bone mineralisation ↑ / Bone pain ↓	Arrhythmias / cramps / PTH ↑
HCO_3	↑	Acidosis control ↑	Postdialytic alkalosis
	↓	Postdialytic alkalosis ↓	Promotes acidosis

Kotanko et al Ch 83 Hemodialysis, Adaquacy and Outcomes 953-966
Comprehensive Clinical Nephrology 3rd Ed Mosby Elsevier 2007
Pastan et al NEJM 1998;338:1428-1436

Dialysis Buffer

- HCO_3-containing solutions were used as the dialysate during the early development of hemodialysis techniques *[KOLFF et al.: The artificial kidney. Acta Med Scand 117:121, 1944]*
- This required the cumbersome aeration of dialysate with CO_2 to prevent precipitation of calcium salts.
- To simplify the hemodialysis procedure, **Mion et al [1964]** introduced the use of **ACETATE** as a source of bicarbonate in dialysate solutions

MI0N CM, HEGSTROM RM, B0EN ST. SCRIBNER BR: Substitution of sodium acetate for sodium bicarbonate in the bath fluid for hemodialysis. Trans Am Soc Artif Intern Organs 10:110, 1964

Disadvantage of HCO_3

- precipitation with calcium and magnesium
- the risk of bacterial contamination,

  bicarbonate was rapidly abandoned and replaced by acetate during the first two decades of dialysis therapy

http://www.kidney-international.org

see commentary on page 909

Survival advantage of hemodialysis relative to peritoneal dialysis in patients with end-stage renal disease and congestive heart failure

Florence Sens[1,2], Anne-Marie Schott-Pethelaz[2,3], Michel Labeeuw[1,3], Cyrille Colin[2,3] and Emmanuel Villar[1,4], on behalf of the REIN Registry

[1]Department of Nephrology, Hospices Civils de Lyon, Lyon-Sud University Hospital, Pierre Benite, France; [2]Pole IMER des Hospices Civils de Lyon, Lyon, France; [3]University Lyon I, Villeurbanne, France and [4]UMR 5558, University Lyon 1, CNRS, Equipe Biostatistiques Santé, Villeurbanne, France

Thus, mortality risk was higher with PD than with HD among incident patients with end-stage renal disease and congestive heart failure. These results may help guide clinical decisions and also highlight the need for randomized clinical trials.

Kidney International (2011) **80,** 970–977; doi:10.1038/ki.2011.233; published online 20 July 2011

Annals of Internal Medicine

Annals of Internal Medicine

Established in 1927 by the American College of Physicians

Less Dialysis-Induced Morbidity and Vascular Instability with Bicarbonate in Dialysate

U. GRAEFE, M.D.; J. MILUTINOVICH, M.D.; W. C. FOLLETTE; J. E. VIZZO; A. L. BABB, Ph.D.; and B. H. SCRIBNER, M.D., F.A.C.P.

Ann Intern Med March 1, 1978 88:332–336;

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Ann Intern Med March 1, 1978 88:332-336;

Abstract

We devised three protocols to test the postulate that increased morbidity during high-efficiency dialysis with large-surface-area units (LS) might be due in part to the increased flux of bicarbonate out and acetate into the patient inherent in LS dialysis. The first protocol showed that with LS-acetate dialysis there was a marked fall in plasma bicarbonate and PCO_2 during the first 3 to 4 h, followed by a rapid rise in bicarbonate above normal and return to control in PCO_2. With LS-bicarbonate dialysis, these oscillations were largely eliminated. A second double-blind protocol showed that central nervous system-type symptoms noted during and after LS-acetate dialysis were reduced significantly by switching to LS-bicarbonate dialysis. The third protocol showed that with LS-bicarbonate the tolerable rate of ultrafiltration could be increased 67% compared with LS-acetate dialysis.

Mechanisms by which Acetate Buffer Contributes to Hemodynamic Instability

- Directly decreases peripheral vascular resistance (in approximately 10% of patients)
- Stimulates release of the vasodilator compound, interleukin 1
- Induction of metabolic acidosis through bicarbonate loss through the dialyzer
- Associated with arterial hypoxemia and increases in oxygen consumption
- Possible Myocardial effects of acetate

Disadvantages of Acetate Dx

- only around 10% of hemodialyzed patients present a severe problem when dialyzed against acetate and should be dialyzed
- against bicarbonate;
- dialysis against acetate does not fully correct the metabolic acidosis even in "normal" patients.

Patrick Vinay et al. Kidney Int 31: 1194-1204; 1987

Proc. EDTA (1979) Vol. 16

THE INFLUENCE OF ACETATE VERSUS BICARBONATE ON PATIENT SYMPTOMATOLOGY DURING DIALYSIS

K Nagai, M Pagel, T Rattazzi, J Vizzo, B H Scribner

University of Washington, Seattle, WA, USA

Patients experienced significantly more symptoms and deterioration of objective performance test scores with both LS–A and LS–C than LS–B. Furthermore, a correlation was seen between plasma acetate level at the end of dialysis and decrement in the performance test scores. The results suggest that accumulation of acetate rather than acute alteration in acid-base status is primarily responsible for the morbidity.

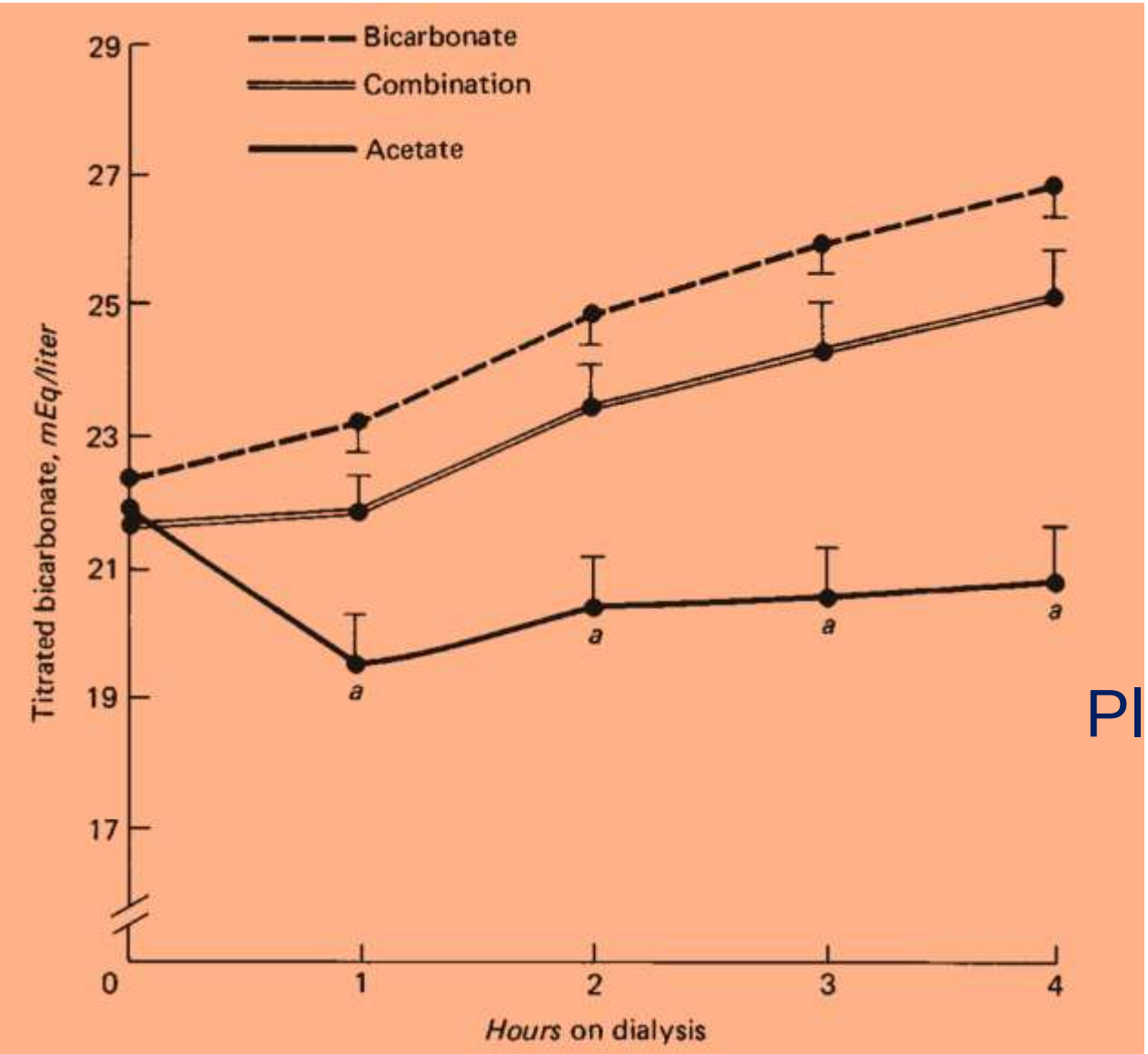
. *Kidney International, Vol. 21 (1982), pp. 513–518*

Acetate and bicarbonate fluctuations and acetate intolerance during dialysis

- 21 stable maintenance dialysis patients undergoing treatments
- Each patient was dialyzed three times – Once each with:
  - 38 mEq/liter acetate (A),
  - 35 mEq/liter bicarbonate (B),
  - and a combination bath containing 38 mEq/liter acetate and 10 mEq/liter bicarbonate (C)
- Each dialysate bath contained 3.5 mEq/liter Ca, 140 mEq/liter Na, and K as required. The total Cl was 102, 105, and 92 mEq/liter for the A, B, and C dialysates, respectively

Pagel et al. *Kidney International, Vol. 21 (1982), pp. 513–518*

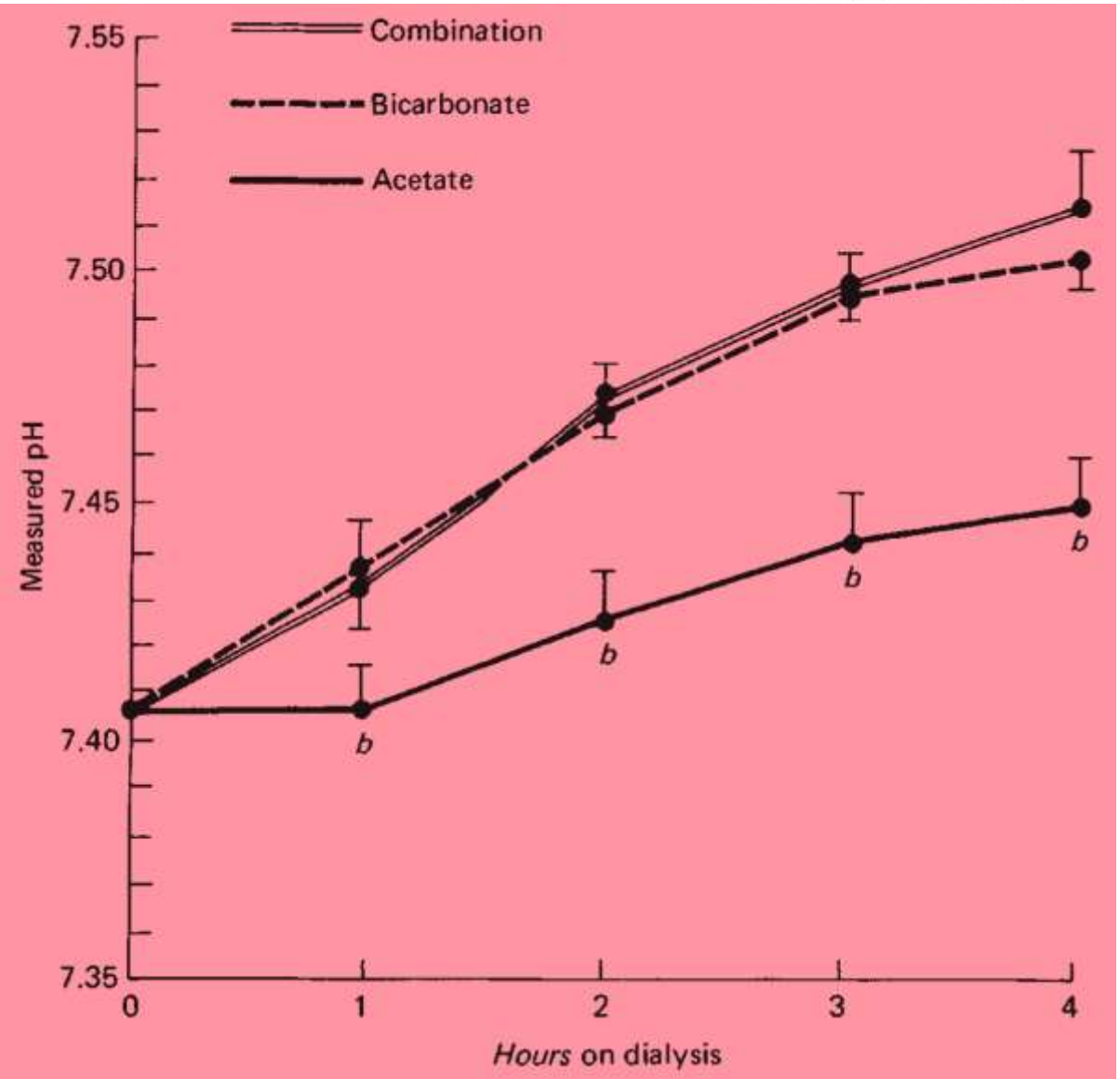
Acetate and bicarbonate fluctuations and acetate intolerance during dialysis



Plasma bicarbonate changes during dialysis

Pagel et al. *Kidney International, Vol. 21 (1982), pp. 513–518*

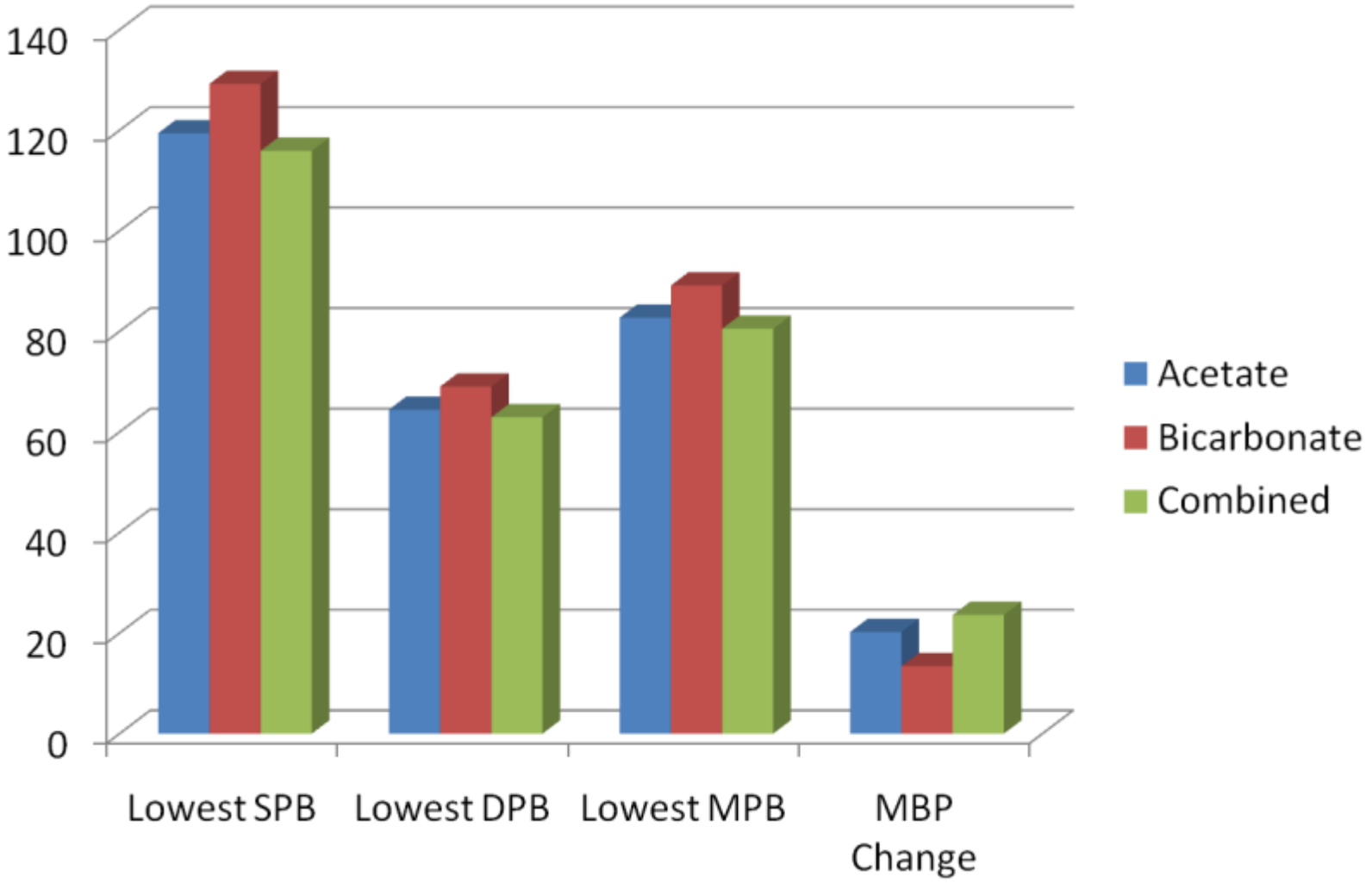
Acetate and bicarbonate fluctuations and acetate intolerance during dialysis



Changes in blood pH during dialysis

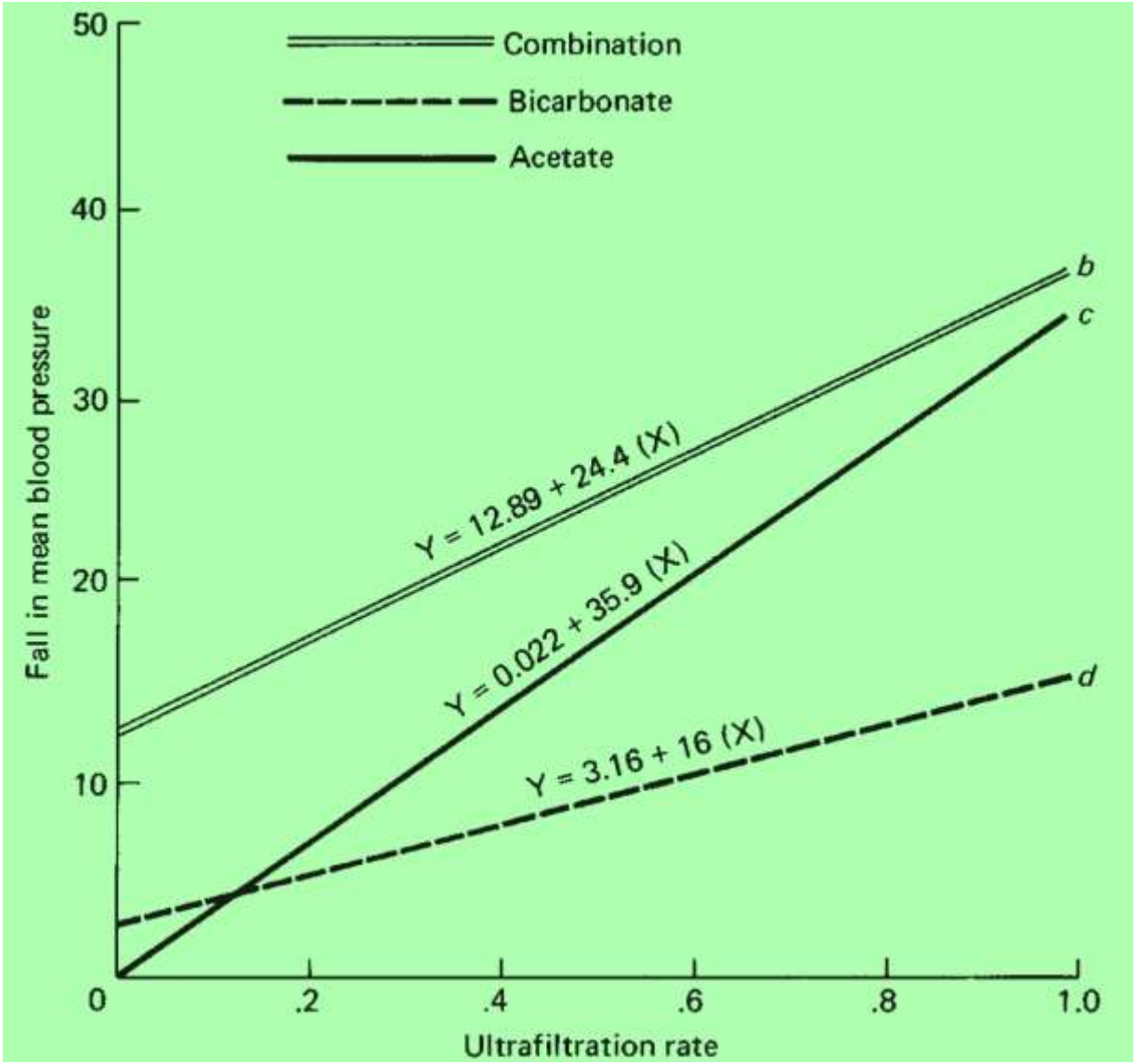
Pagel et al. *Kidney International, Vol. 21 (1982), pp. 513–518*

Acetate and bicarbonate fluctuations and acetate intolerance during dialysis



Pagel et al. *Kidney International, Vol. 21 (1982), pp. 513–518*

Acetate and bicarbonate fluctuations and acetate intolerance during dialysis



Regression line of fall in mean blood pressure versus rate of ultrafiltration

Pagel et al. *Kidney International, Vol. 21 (1982), pp. 513–518*

Acetate and bicarbonate fluctuations and acetate intolerance during dialysis

Symptoms and performance task scores during dialysis

Frequency of symptoms	Acetate (A)	Bicarbonate (B)	Combination (C)
Nausea	10	2	10
Headache	11	11	14
Vomiting	2	0	1
Total	23[a]	13	25[b]
Reaction time change, *msec*[c]	29.3[a] ±38.1	9.1 ±37.1	40.9[b] ±41.8

[a] A vs. B, $P < 0.05$.
[b] C vs. B, $P < 0.01$.

Kidney International, Vol. 24 (1983), pp. 240–245

High sodium bicarbonate and acetate hemodialysis: Double-blind crossover comparison of hemodynamic and ventilatory effects

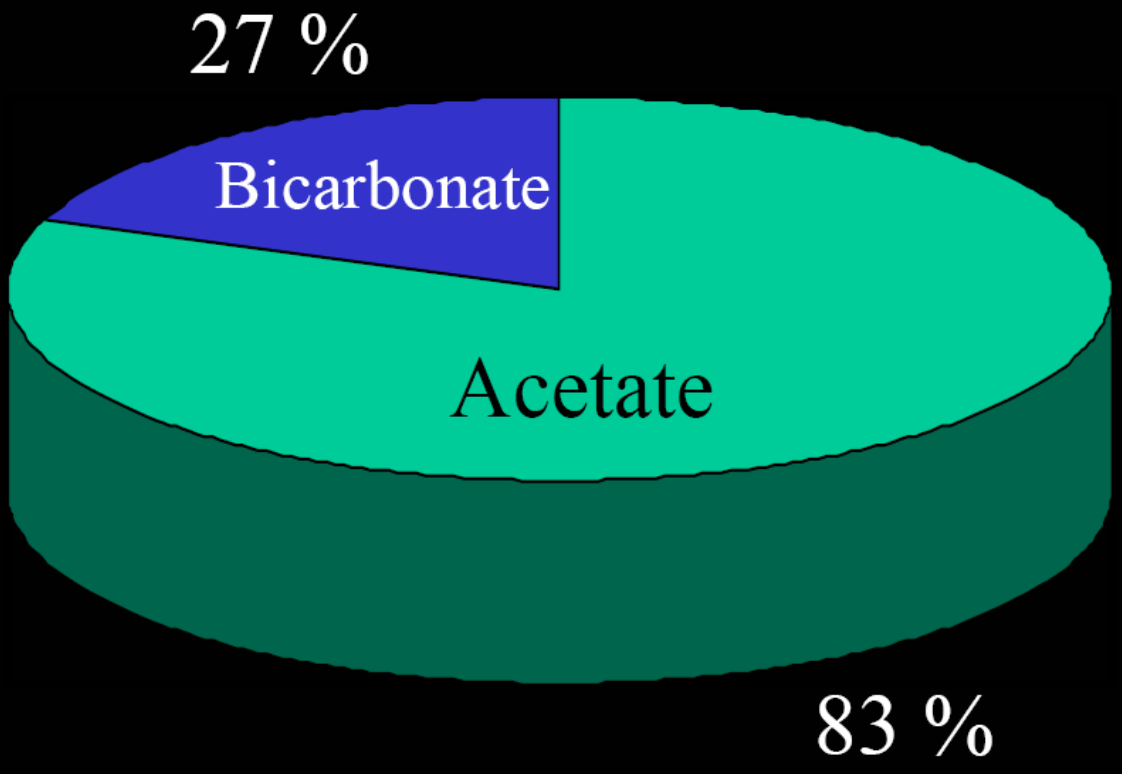
WILLIAM L. HENRICH, TERRY D. WOODARD, BARRY D. MEYER, TIMOTHY R. CHAPPELL, and LEWIS J. RUBIN

Department of Internal Medicine, Divisions of Nephrology and Pulmonary Disease, University of Texas Southwestern Medical School and Dallas Veterans Administration Medical Center, Dallas, Texas

In summary, these results demonstrate strikingly similar hemodynamic and ventilatory responses with the two dialysates when a higher osmolality dialysate is used. However, Bi HD was associated with a significant reduction in the number of therapeutic interventions required, and also resulted in a greater pre-HD pH and bicarbonate concentration.

Hemodialysis Solutions

n. 3415



Adel Affifi: The Egyptian Renal Registry 9th Annual Report 2008

HAEMODIALYSIS CONCENTRATED SOLUTION

Dilution Ratio 1 : 34
(B.P 2003)

ACETATE FORMULA

M.O.H Reg. No. : 970 / 2008

NON-PYROGENIC

Formula for 1 L of diluted solution

Na^+	138	mEq.	Mg^{++}	1.5	mEq.
K^+	1.5	mEq.	Cl^-	107	mEq.
Ca^{++}	2.5	mEq.	$CH_3\ Coo^-$	36.6	mEq.

INSTRUCTIONS FOR USE

- Concentrated Solution is to be diluted immediately Before use
- Volume taken for use is to be measured accurately
- Any unused portion of solution is to be discarded
- Store at a temperature not below 4C

Volume : 20 L
Batch No. : AC 6-11
Manf.Date : 5/2011
Exp. Date : 5/2 13

EL MANSOURA CO. FOR MEDICAL AND CHEMCAL INDUSTRIES

MADE IN EGYPT

Head office : El Mansoura 153 Suiz Canal St.Borg El kheir Hosp.
Factory : Gamasa 1 Industrial Area Block 17 , 18 H Chemicals

شركة المنصورة للصناعات الطبية والكيماوية

صنع في مصر

TEL +2 050 2339823-23-25
TEL +2 050 2770011

HAEMODIALYSIS CONCENTRATED SOLUTION

Bicarbonate Formula (B.P 2003)

Dilution Ration (1:35.2)

(Compenent A)

Each Plastic Container 20 Liters Contain:		
Glacial Acetic Acid	135.8	ml
Sodium Chloride	4.442	kg
Potassium Chloride	108	gm
Calcium Chloride	186	gm
Magnesium Chloride	73.6	gm

Ionic Formula per liter		
Na^+	105	mmol/L
K^+	2	mmol/L
Ca^{++}	1.75	mmol/L
Mg^{++}	0.5	mmol/L
Cl^-	111.5	mmol/L
$CH_3 Coo^-$	3.0	mmol/L

BICARBONATE

M.O.H Reg No. 9717/2008

NON-PYROGENIC

INSTRUCTIONS FOR USE

- The concentrated soution is to be diluted immediately before use. Store at atemperature not below 4c .
- The volume taken for use is to be measured accurately.
- Unused portion of solution is to be discarded.
- Component (b) is to be added before use.

Volume : 20 L
Batch No. : 31-17-1
Manf.Date : 2
Exp. Date : 0

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Factory : Gamasa 1 industrial Area Block 17 , 18 H Chemicals

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صنع في مصر

TEL +2 050 2339823-23-25
TEL +2 050 2770011

SODIUM BICARBONATE POWDER B.P. 2001
For Bicarbonate Haemodialysis

بيكربونات صوديوم ف.ب. ٢٠٠١

خاص بمحلول الكلى الصناعى المركز (بيكربونات)

الوزن الصافى : ٦٥٠ جم

طريقة الإستعمال : تذاب هذه الكمية فى ٨ لتر ماء مقطر
مكونات (جزء ب / Component B) الذى يستعمل مع ٨ لتر
من محلول الكلى المركز (جزء أ / Component A)

ويحظر الإستخدام منفردا

Warning :

- This packet can not be used by itself for Haemodialysis, it should be used with Component A.
- Bicarbonate solution must be used in the same day of preparation.
- Keep tightly sealed until used avoiding excessive heat.

Batch No. : 2011/10
Manf. Date : 3/2011
Exp. Date : 3/2016

النصر للكيماويات الداوئية
أبو زعبل مصر
Made in Egypt


A
B


A
B


B
B


BiCart®
Sodium bicarbonate (NaHCO3, Ph. Eur., USP)
EN For dialysis
ES Para diálisis
DE Für die Dialyse
FR Pour dialyse
CS Pro dialýzu
DA Til dialyse
EL Για αιμοδιάλυση
ET Dialüüsi jaoks
FI Dialyysiä varten
HU Dializis céljára
IT Per dialisi
LT Dializei
LV Dialīzei
NL Voor dialyse
NO For dialyse
PL Do dalizy
PT Para diálise
SK Pre dialýzu
SL Za dializo
SV För dialys
DIN 00882666. Patent US 4,784,495.
CE 0086
B10640 Rev. 2005-02
Exp. date


Tom medical
B
Hemodialysis bicarbonate cartridge
NaHCO3 Eur.Ph
720g
Tom Medical Supplies Co.,Ltd.
Http://www.tmsplc.com
ersser.en.alibaba.com
31.05.2011 11:37

JK SCIENCE

SHORT ARTICLE

Vol. 1 No. 1 January, 99

Metabolic Acidosis as a Complication of Bicarbonate Haemodialysis

Irshad Ahmad Sirwal, Bassam Bernieh, Abdulrahman Osman Mohamad, Mohamed Adnan Abbadi, Mossadeque Ahmed, Ahmad Abdelwahab Altabakh

From the Department of Nephrology, King Fahad Hospital, Medinah Al Munawara~ KSA.

12 episodes of severe metabolic acidosis were observed among 10 maintenance dialysis patients using Bicarbonate Haemodialysis (HDB). Patients were stable at the start of haemodialysis (HO) and became sick during or following the procedure. The main clinical features observed were abdominal pain and vomiting, hypotension or shock, and CNS manifestations. Laboratory investigations revealed severe metabolic acidosis in all and hyperkalemia in 4 patients. On four occasions, dialysate fluid sample analysis revealed purely acidic dialysate being delivered to the patients. Patients were treated by sodium bicarbonate, redialysis on another machine and vasopressors when severely hypotensive. One patient died and the rest improved. This potentially lethal complication needs to be considered early in all patients who become sick during or following HDB.

JK SCIENCE

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The use of acetate and bicarbonate dialysis at the same dialysis centre results in the necessity of use of different dialysate concentrates. Hence, there is an increasing chance of mistake by using the wrong concentrate. Dialysis equipment can proportion the dialysis fluids using an acid concentrate as acetate and still obtain the proper conductivity without setting off alarms. Mixture of only the acidic component with water may not be detected by conductivity-meter and unless a pH meter is included, an acid dialysate will be delivered to the patient inducing life threatening metabolic acidosis.


CITRASATE
most prescribed
citric-acid
concentrate

Citrasate® Acid Concentrates

45x	Product Code Number	Na+ mEq/L	K+ mEq/L	Ca++ mEq/L	Mg++ mEq/L	Cl- mEq/L	Acetate mEq/L	Dextrose mg/dL	Citrate mEq/L
Case = 4 Bottles 1 bottle = 1 gallon (3.78 Liters)	08-1251-CA	100.3	1	2.50	1.00	104.50	0.3	100	2.4
	08-2251-CA	100.3	2	2.50	1.00	105.50	0.3	100	2.4
	08-3251-CA	100.3	3	2.50	1.00	106.50	0.3	100	2.4
1 Drum = 55 gallons (208.2 Liters)	13-1251-CA	100.3	1	2.50	1.00	104.50	0.3	100	2.4
	13-2251-CA	100.3	2	2.50	1.00	105.50	0.3	100	2.4
	13-3251-CA	100.3	3	2.50	1.00	106.50	0.3	100	2.4

Acid concentrate formulas are expressed as acid portion only prior to the addition and reaction of Fresenius sodium bicarbonate. For use with hemodialysis equipment capable of calibration for a mix ratio of 1:44 (also expressed as 45x or 1:1.72:42.28)

NxStage System One: Treatment when you want, how you want and where you want

Thanks